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ORIGINAL ARTICLE

Radiofrequency catheter ablation of premature ventricular complexes from right ventricular outflow tract in patients with left ventricular dilation and/or dysfunction

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KEYWORDS

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Abstract Left ventricular (LV) dysfunction caused by frequent premature ventricular complexes (PVCs) can be reversed by suppression of PVCs with antiarrhythmic agents or radiofrequency catheter ablation (RFA). However, there is a paucity of data on the efficacy and safety of RFA among the local population. We aimed in this study to evaluate the effect of RFA of frequent PVCs originating from right ventricular outflow tract (RVOT-PVCs) on cardiac function in patients with depressed cardiac function and/or LV dilation. The study included sixteen patients with monomorphic RVOT-PVCs without overt underlying structural heart disease. Frequency of PVCs by 24-h Holter monitoring, left ventricular ejection fraction (LVEF), end-diastolic diameter (LVEDD), end-systolic diameter (LVESD), mitral regurgitation (MR) by echocardiogram and NYHA functional class were evaluated before and 3 and 6 months after RFA. All patients underwent RFA.
Results: The higher the number of PVCs/24 h, the bigger the LVESD and the lower the EF. Procedural success was achieved in 13 (81%) of the patients with no complications. Six months follow-up after successful ablation, LVEDD decreased significantly (from 56.62 ± 5.87 to 49.23 ± 5.31 mm; $p = 0.002$), LVESD decreased significantly (from 41.85 ± 7.82 to 33.69 ± 4.66 mm; $p = 0.002$), LVEF increased significantly (from 46.69 ± 4.92% to 60.54 ± 5.39%; $p < 0.001$) and NYHA functional class improved in all patients after successful ablation.

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Conclusion: RF catheter ablation of frequent RVOT-PVC has a beneficial effect on cardiac function in patients with depressed cardiac function.
It carries a high degree of success and safety. Frequent RVOT-PVCs are burden on LV function even in patients without overt underlying structural heart disease.

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1. Introduction

Premature ventricular contractions (PVCs) are early depolarization of the myocardium originating in the ventricle. They are often seen in association with structural heart disease and represent increased risk of sudden death, yet they are ubiquitous, even in the absence of identifiable heart disease.[1,2] They may cause troubling and sometimes incapacitating symptoms such as palpitation, chest pain, presyncope, syncope, and heart failure.[3] Idiopathic ventricular arrhythmias (VA) consist of various subtypes of VA that occur in the absence of clinically apparent structural heart disease. Affected patients account for approximately 10% of all patients referred for evaluation of ventricular tachycardia (VT).[4] Arrhythmias arising from the outflow tract (OT) are the most common subtype of idiopathic VA and more than 70–80% of idiopathic VTs or (PVCs) originate from the right ventricular (RV) OT.[5] Traditionally, PVCs have been thought to be relatively benign in the absence of structural heart disease,[6] but in retrospective analyses premature ventricular complexes have been implicated as a cause of impaired left ventricular function.[2] Also frequent isolated ectopic beats, mostly originating from the right ventricular outflow tract have been reported as a cause of tachycardiomyopathy, a reversible form of congestive heart disease that resolves after elimination of the culprit arrhythmia either by medical treatment or by Radiofrequency Ablation (RFA).[7] Various therapeutic options for ventricular arrhythmias include antiarrhythmic drugs, antiarrhythmic surgery and placement of an implantable cardioverter defibrillator (ICD) for VT, or radiofrequency catheter ablation.[8] When highly symptomatic and refractory to antiarrhythmic therapy or causative for ventricular dysfunction, ablation is a recommended treatment for right ventricular outflow tract PVCs (RVOT-PVCs) with a high success rate and a low risk of complications.[9]

2. Aim of the work

This study was undertaken to evaluate the effect of RF ablation of frequent premature ventricular complexes from right ventricular outflow tract (RVOT-PVCs) on cardiac function in patients with depressed cardiac function and to examine whether frequent RVOT-PVCs without overt underlying structural heart disease correlates with LV dilation, which is a well-recognized precursor of LV dysfunction and congestive heart failure or not.

3. Patients and methods

The study was conducted at the Cardiology department of our institute from February, 2011 to September, 2013. Patients enrolled in this study when they have symptomatic monomorphic RVOT-PVCs associated with LV dilation (LVEDD $\geqslant$59 mm and/or LVESD $\geqslant$41 mm) and/or impaired LV systolic function (EF $\leqslant$50%) without overt underlying structural heart disease. RVOT-PVC was defined as a characteristic electrocardiographic appearance of a left bundle branch block (LBBB) contour in V1 and an inferior axis in the frontal plane. We excluded patients with the history of recent myocardial infarction, patients with ECG evidence of myocardial infarction, patients with known coronary stenosis of $>$50% diameter, patients with segmental dyskinetic regions by Echocardiography, patients with significant valvular heart disease, patients with congenital heart disease, patients with RV abnormalities, Patients with atrial tachyarrhythmia including AF, atrial flutter, atrial tachycardia, and paroxysmal supraventricular tachycardia and any patient unwilling to have the procedure. LVOT-PVCs diagnosed according to Betensky's algorithm,[10] which utilized the 12-leads surface ECG for PVC localization, were excluded from the study.

All patients signed an informed consent after discussing the procedures and possible alternatives and complications. All patients were subjected to through clinical examination, history taking and routine laboratory analysis. Chest radiogram in PA view was done for the measurement of CT ratio and detection of cardiomegaly. Resting 12-Leads surface ECG was performed to detect morphology of PVCs (if frequent) and to exclude evidence of myocardial infarction or evidence of other arrhythmia. Resting Trans-Thoracic Echocardiography (TTE) was undertaken (with the patient lying in the left lateral decubitus using the standard views) on admission, 3 and 6 months after the successful RF ablation. From TTE we can estimate chamber size of LV and right ventricle (RV), Degree of mitral regurgitation (at parasternal long-axis or apical four-chamber view) and Left ventricular ejection fraction (LVEF) which was calculated using the Teichholz method. All values of echocardiogram were recorded during sinus rhythm, but not at the PVC beat, nor at the post-PVC beat. All patients also had routine echocardiogram on the next day of RFA to evaluate the possible procedure related complications. 24-h Holter monitoring was done to all patients before RFA, within 48 h after RFA, 3 and 6 months thereafter and whenever there are significant symptoms during follow up period to exclude recurrence. The %PVC was calculated as: 100·[number of PVC/number of total heart beats per 24 h]. Coronary angiography (CAG) was done for high risk patients to exclude ischemic heart disease, and exercise stress test (ETT) was done for low and intermediate risk patients for the same purpose.

Electrophysiological study, mapping and catheter ablation were done by the conventional method. The patients were studied in the fasting state without sedation. Antiarrhythmic drugs were discontinued for at least six half-lives before the procedure. Under local anesthesia, two 7-F deflectable quadripolar ablation catheters with a 4-mm-tip electrode and 5 mm interelectrode distance were introduced percutaneously into the RV. One of the two catheters was used first to make a land mark for the His position. The ablation catheter was introduced to the RVOT guided by both LAO and RAO

projections. Induction of VT was tried in patients when their history is suggesting VT or if they have a documented VT (either sustained or non-sustained) recorded by 12-leads ECG or during Holter monitoring. Mapping procedure was started by looking for the earliest local ventricular activation of PVCs 20–60 ms before the onset of QRS complex in V1 of the simultaneously recorded ECG. Roving technique, sometimes was used by the two ablation catheters, the first catheter was moved to find the earliest ventricular activation site and the second ablation catheter was moved to find an earlier ventricular activation than the first. This maneuver was repeated until the earliest ventricular activation site of PVCs was found (Fig. 1). Bipolar pace mapping (of 2 ms pulse width at double the diastolic threshold) was used immediately before applying RF current at the culprit site of ablation in order to match the 12 leads ECG obtained during pacing with the spontaneous PVCs of the patient. An identical match was necessary in at least 11 of 12 leads.

Epinephrine administration and/or programmed electrical stimulation was performed to induce the culprit PVC. If the culprit PVCs were infrequent at the time of the study a combination of activation and pace mapping was used to identify the optimal target site of ablation in order to prevent prolongation of procedure time. RFA was delivered for 60–90 s at the site that poses an identical paced 12-lead ECG match.

During mapping for earliest ventricular activation RFA was also performed for 60–90 s. If the culprit PVCs did not disappear after the 10 s ablation stopped searching for another site was attempted.

A successful ablation was defined as no recurrence and non-inducibility of culprit PVC with and without programmed electrical stimulation for at least 30 min after ablation. All 12-leads surface electrocardiograms and the bipolar intracardiac electrograms (filtered at 30–400 Hz) were recorded and stored by using the channel acquisition system. During the procedure, intravenous heparin was given as a 70 IU/kg bolus dose followed by

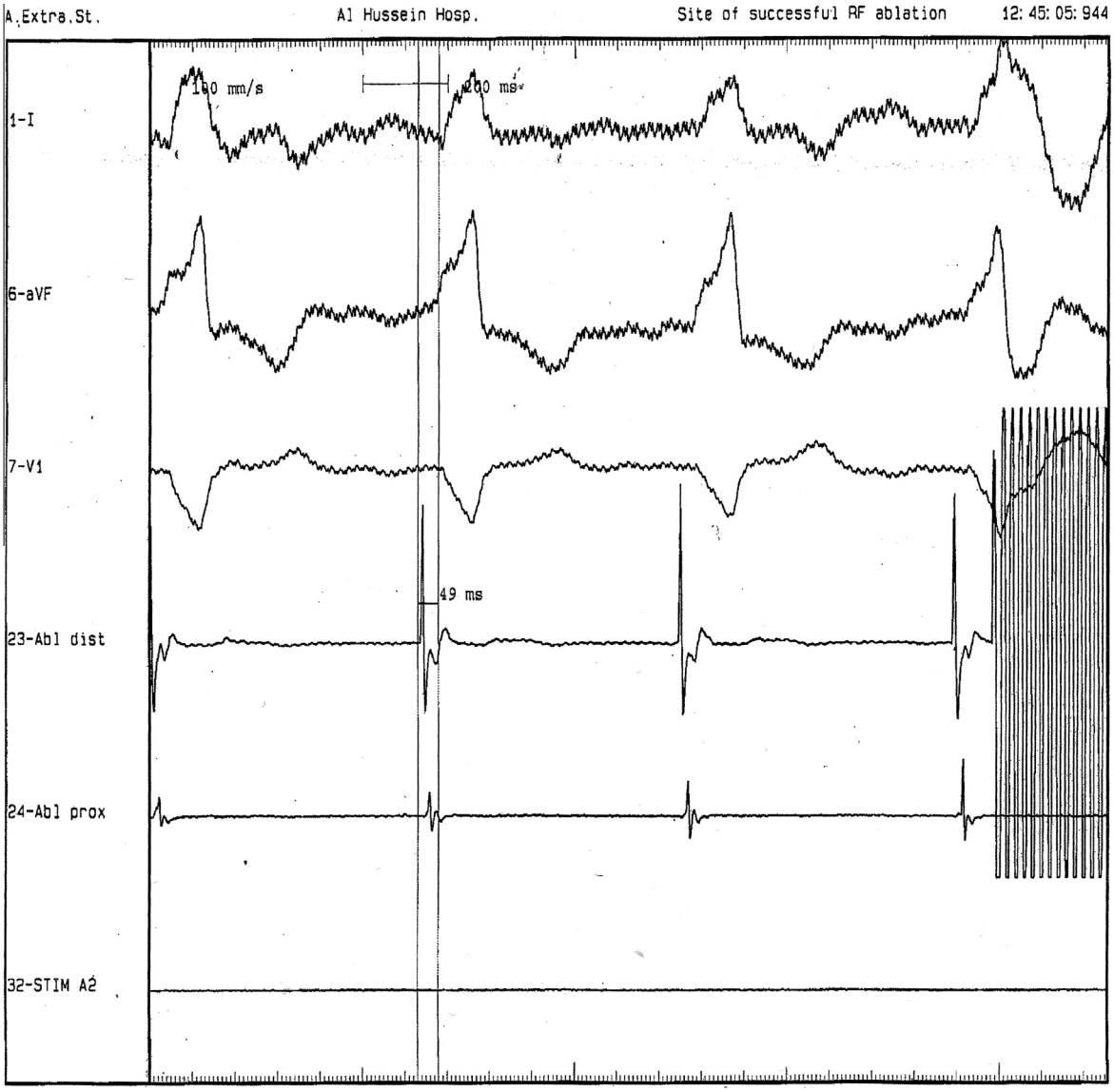

Figure 1 Shows earliest ventricular activation site at the ablation catheter 49 ms before culprit PVC. ECG leads I, aVF, V1 and intracardiac recording by the ablation catheter.

boluses of 1000 IU every hour. All patients received oral antiplatelets for 6 weeks after RFA, but no antiarrhythmic drugs.

The standard of instant success is as follows: When PVCs disappear or when sporadic PVCs ⩽1 beats/min, VT cannot be induced after radiofrequency ablation, when close observation for 30 min after procedure reveals a reduction of the total number of PVCs to less than 10 (shape is completely similar to monomorphic PVCs before procedure).[11]

It should be confirmed in all cases that the PVC was not caused mechanically and that it coincided with the morphology of the clinical PVC. An increase in ectopic activity may be observed to precede termination of the application. Sometimes, at successful ablation site when applying RF, acceleration of tachycardia may occur before its termination.

The standard of long-term success is when Holter ECG monitoring for a full day within 3 months after successful procedure shows that PVCs disappeared, or when total number of PVCs decreased by over 75%. Moreover, long-term success is achieved when the uncomfortable symptoms are remarkably improved.[11]

4. Statistical analysis

Clinical and electrophysiological variables were recorded and a descriptive analysis was done. Data were collected on a special format, verified and then coded when needed prior to analysis. All continuous data were expressed as mean ± SD unless otherwise needed, categorical data were expressed as frequency in tables. Enumeration data are described as case numbers and percentages. Chi-square test for assessing association in categorical data was performed. p-value <0.05 was considered significant. Nonparametric tests were used for correlation between different variables. All analysis had been performed using ***Statistical Package for Social Sciences*** (SPSS) version 10 for Windows and graphics by MS Excel.

5. Results

Sixteen patients fulfilled the inclusion criteria described previously in the methodology, baseline characteristics of the patient population are summarized in Table 1. Three patients with high risk of IHD had normal coronary angiography. Thirteen patients with low risk of IHD were subjected to exercise test, two of them developed short runs of VT during exercise that required termination of the test and referred for CAG which was found normal. PVCs frequency increased in five patients and disappeared in the rest of patients at peak exercise.

The age ranged from 13 to 53 years and the mean age was 34 ± 12, the total number of females was 11(69%). All patients included in the study had PVCs-associated symptoms, seven patients were complaining of Dyspnea, six patients from syncope and two patients from palpitation while the remaining patient was complaining of chest pain. All patients had been taking antiarrhythmic agents before admission. The duration of symptoms in months for each patient was reported. It ranged from 12 to 46 months and the mean duration was 25.13 ± 11.43. Eleven patients (69%) were in NYHA functional class II and five patients (31%) in class III. Detailed mapping of the right ventricular OT was performed in the 16 patients. In 1 patient ventricular ectopy was infrequent and required intensive pace mapping with searching for an identical paced 12-leads ECG match. None of the patients had absent PVCs at the time of the study. Procedural acute success was achieved in 13 patients (81%) of the 16 patients, and no procedure-related complications were observed in all studied patients. Procedural success of the patient with infrequent PVCs was documented by the disappearance of these PVCs following ablation even with isopretrenol infusion, this disappearance was assured by Holter recording 48 h after ablation.

The site of successful ablation was found to be septal in ten (62%) patients, free wall in three (19%) patients but not localized in the remaining three patients (19%) with failed RFA. The average procedure time was 118.12 ± 50.42 (35.00–200.00) min beginning from puncturing to removal of the sheath. The average *Watts* used for ablation is 33.44 ± 2.39 (30.00–35.00). All patients were followed up for a mean period of 13.25 ± 7.8 (6–30) months. During follow-up there was significant reduction in the number and percentage of culprit RVOT-PVCs in all patients with acute success for 48 h, 3 and 6 months after ablation ($p = 0.001$).

The number of PVCs decreased from 25640.50 ± 17461.55/24 h. before ablation to 928 ± 441/24 h. in 48 h after ablation, 892 ± 408/24 h. in 3 months after ablation and 857 ± 389/24 h. in 6 months after ablation. The percentage of PVCs decreased from 21 ± 9.48 before ablation to 0.84 ± 0.4 in the 48 h after ablation, 0.78 ± 0.36 in 3 months after ablation and 0.68 ± 0.32 in 6 months after ablation (Fig. 2).

There was nonsignificant correlation between EDD and percentage or total number of PVCs before ablation (p value = 0.252). There was significant positive correlation between ESD and total number of PVCs before ablation, ($r = 0.490$, $p = 0.01$), (Fig. 3). There was significant positive correlation between ESD and percentage of PVCs before ablation, ($r = 0.476$, $p = 0.015$), (Fig. 4). There was a significant

Table 1 Baseline characteristics of the studied population.

	N	Minimum	Maximum	Mean	Std. D.
Age	16	13.00	53.00	34.63	12.82
Duration of symptoms in months	16	12	46	25.13	11.43
EDD before ablation/mm	16	40	66	57.06	5.34
ESD before ablation/mm	16	24	57	42.81	7.32
EF before ablation	16	34	52	45.19	5.85
Number of PVCs before ablation/24 h	16	8698	82,753	25640.50	17461.55
Total number of heart beats/24 h	16	71,535	212,418	117083.81	29960.03
% of PVCs	16	7.00	39.00	21.31	9.48

EDD, end-diastolic diameter; ESD, end-systolic diameter; EF, ejection fraction; PVC, premature ventricular contraction.

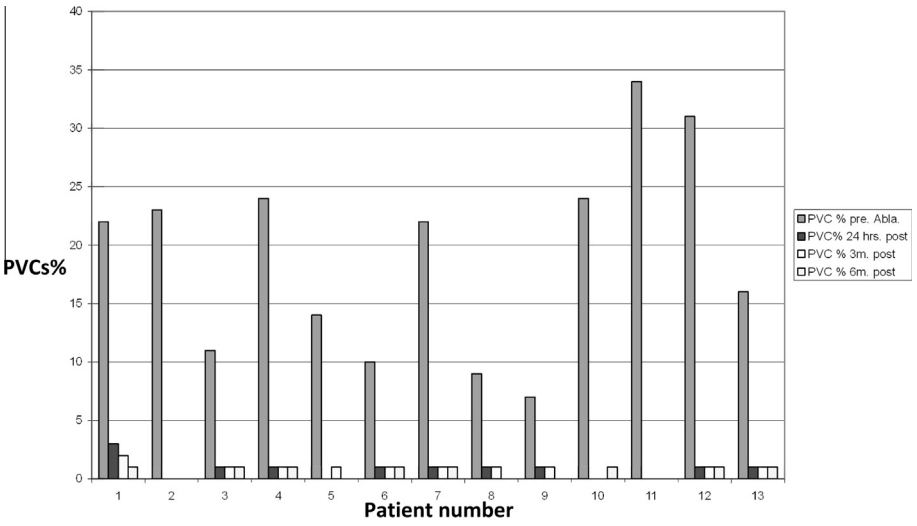

Figure 2 Shows a high significant reduction in PVCs% 48 h, 3 and 6 months following successful RFA when compared with pre-ablation PVCs% ($p = 0.002$).

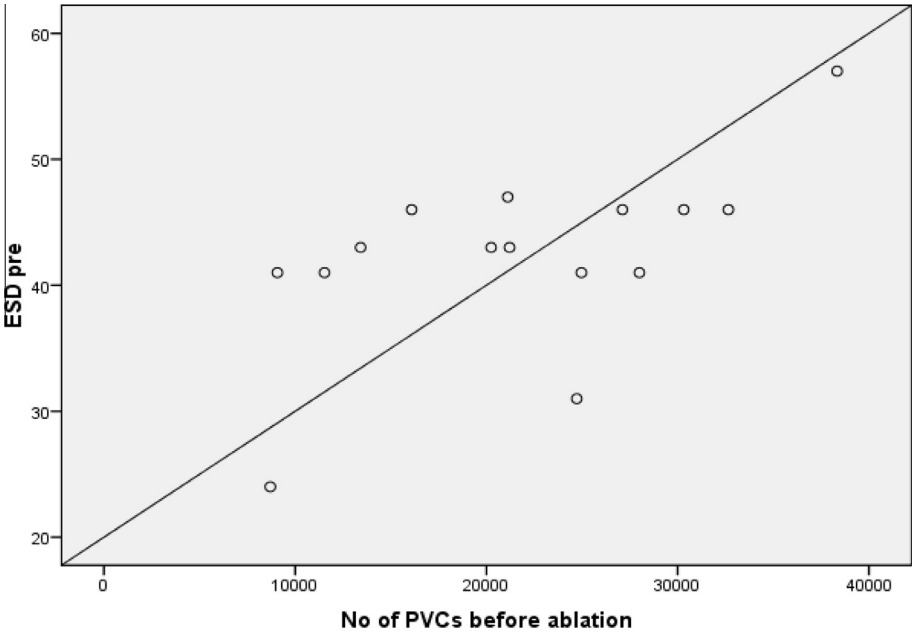

Figure 3 Significant positive correlation between ESD and total number of PVCs before ablation ($r = 0.490$, $p = 0.01$).

inverse correlation between the total number of PVCs and the EF before ablation ($r = -0.587$, $p = 0.002$), (Fig. 5). There was a significant inverse correlation between percentage of PVCs and the EF before ablation ($r = -0.505$, $p = 0.008$), (Fig. 6).

In the thirteen patients with successful RFA, there was a high significant reduction in EDD and ESD 3 & 6 months after ablation when compared with the baseline measures (Figs. 7 and 8), (Table 2) On the other hand, a highly significant improvement was observed in EF in all patients with successful RFA when compared with baseline EF (Fig. 9), (Table 2). NYHA functional class improved in all patients with successful ablation (11 patients were in NYHA class II and 2 in NYHA class III improved to 12 patients in NYHA class 1 and 1 patient in NYHA class II ($p = 0.004$).

Patients with unsuccessful RFA (three patients) did not show any improvement in LVEF or decrease in LVESD in follow up period. Also LVEDD and degree of mitral regurgitation did not show any improvement but sometimes showed deterioration especially in NYHA functional class (Table 3).

6. Discussion

Radiofrequency catheter ablation has become a promising and effective curative procedure for the treatment of symptomatic idiopathic premature ventricular contractions originating from the right ventricular outflow tract in patients without structural heart disease.[9] However, there is a paucity of data on the efficacy and safety of radiofrequency catheter ablation

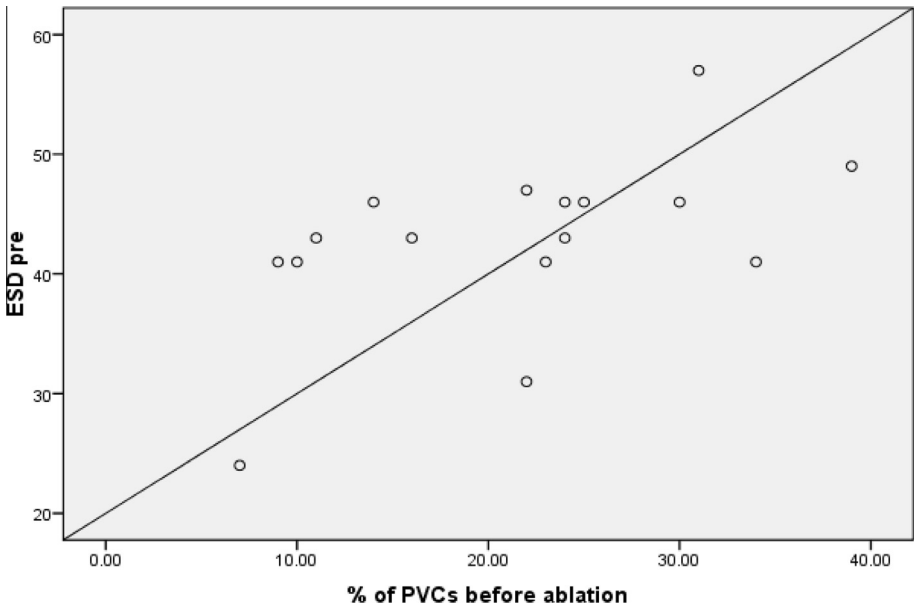

Figure 4 Significant positive correlation between ESD and percentage of PVCs before ablation ($r = 0.476$, $p = 0.015$).

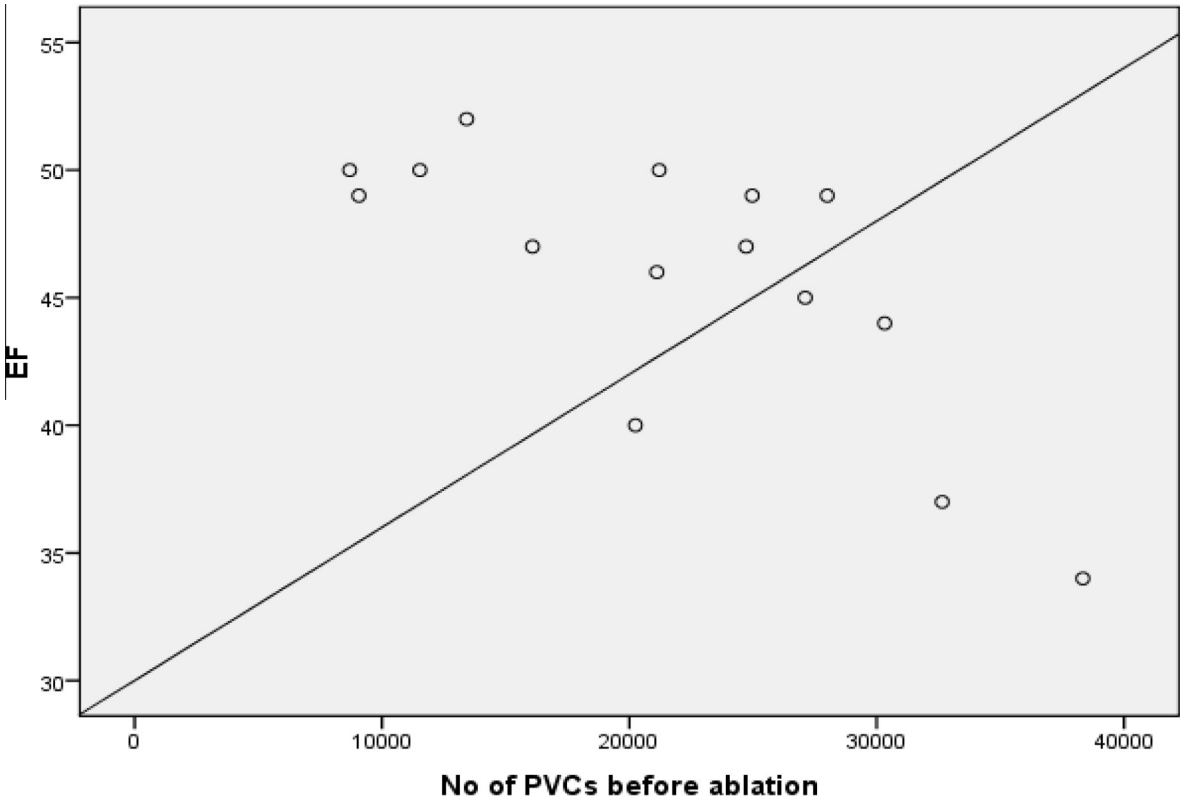

Figure 5 Significant inverse correlation between total number of PVCs and EF before ablation ($r = -0.587$, $p = 0.002$).

among the local population. The objectives of this study were to evaluate the outcome of RFCA as curative therapy for idiopathic RVOT-PVCs and its effect on the ventricular function and left ventricular dimensions. The study also aimed to identify the extent of procedure-related complications. In the present study, RFA procedure for RVOT-PVCs was performed in 16 patients with LV dilation and/or dysfunction without overt underlying structural heart disease. Acute success was achieved in 13 (81%) of the patients, with no procedure related complications. All patients included in our study had PVCs-associated symptoms which were in the order of frequency: dyspnea, syncope, palpitation and finally chest pain. All

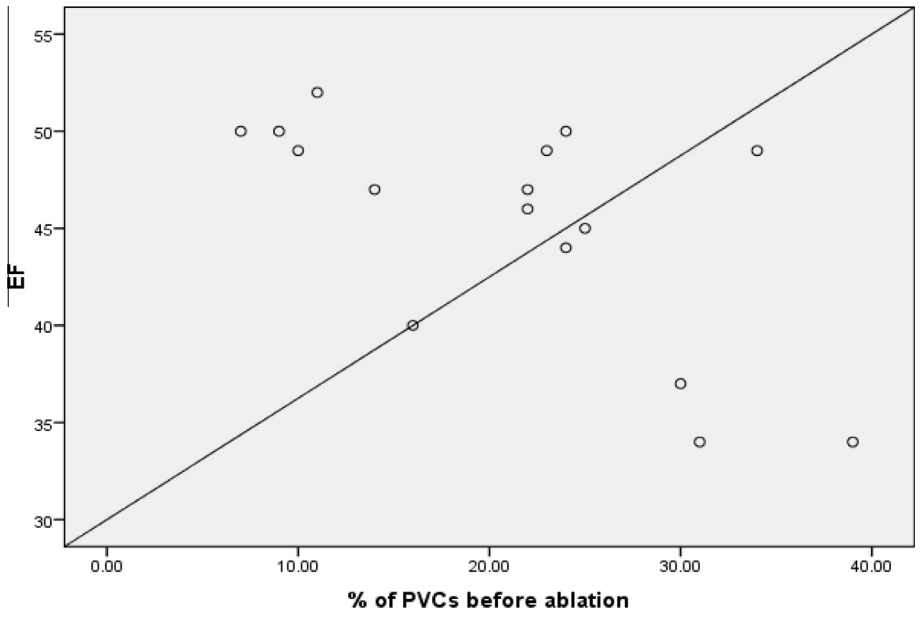

Figure 6 Significant inverse correlation between percentage of PVCs and EF before ablation (r = -0.505, p = 0.008).

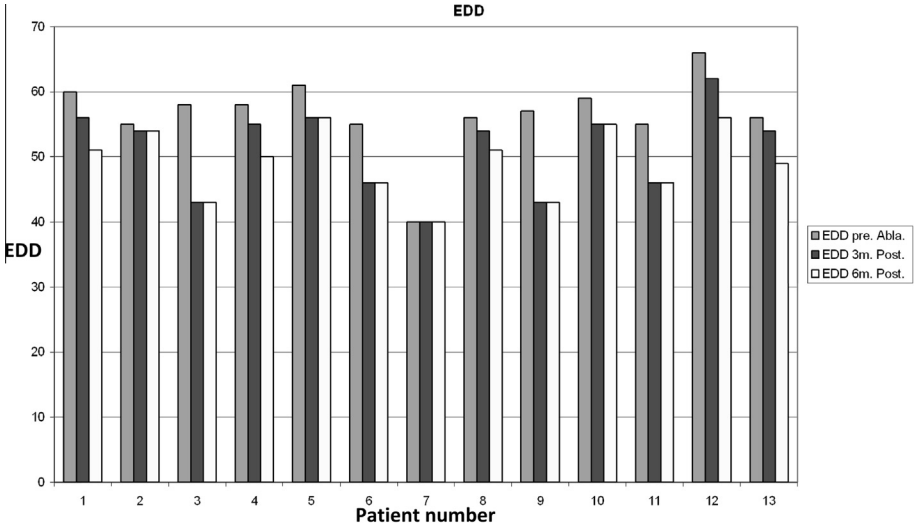

Figure 7 Shows a high significant reduction in EDD 3 and 6 months following successful RFA when compared with pre-ablation EDD (p = 0.002).

patients with successful procedure reported the absence of PVCs associated symptoms and they discontinued the antiarrhythmic agents after RFA. These findings regarding PVCs-associated symptoms are similar to findings of Takemoto et al.; however palpitation was the most frequent symptom in their study.[12]

One patient in our series was complaining of chest pain several years before his inclusion in our study, the history of this patient revealed that he had two diagnostic coronary angiography in 2 years period for the purpose of investigating his chest pain and was found to be normal, however, since this was a subjective main complain of the patient it was mentioned as it was described by the patient. Chest pain in this patient disappeared after successful ablation. On reviewing publications some patients in other series were complaining of chest pain.[3,11–13] The exact cause for chest pain in patients with PVCs is not clear. A decreased effective cardiac output could be an explanation for this chest pain.[3] Animal models have shown that right ventricular pacing induces asymmetrical myocardial hypertrophy, myofibrillar disarray and increased catecholamine concentrations in the myocardium that evoke sympathoexcitation.[14,15] which might be the cause of chest pain in some individuals.

The percentage of acute success in our study was comparable to the experience of other various centers, where success rates have been reported between 75 and 100%.[9] Darrieux et al. achieved 77% success rate[16] while van Huls van Taxis et al. achieved 100% success.[17]

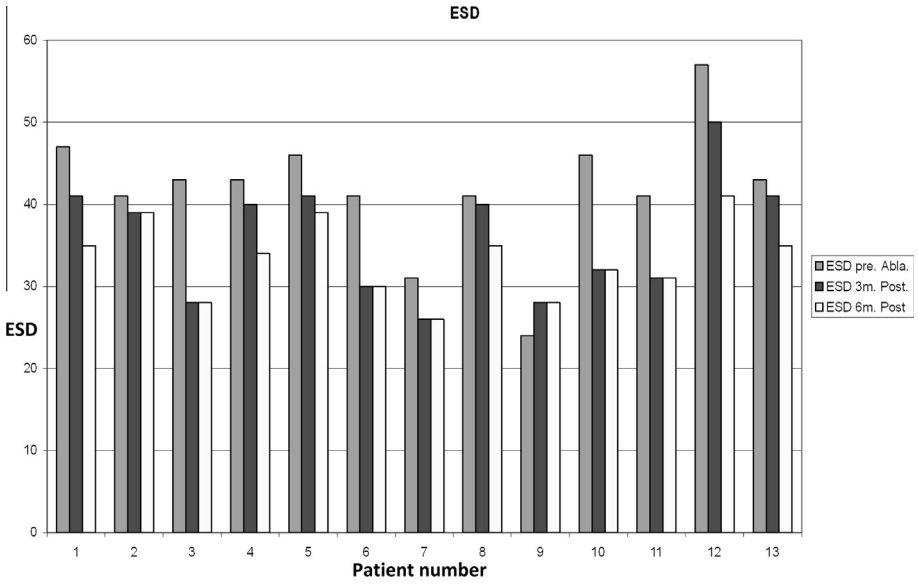

Figure 8 Shows a high significant reduction in ESD 3 and 6 months following successful RFA when compared with pre-ablation ESD ($p = 0.005$ and 0.002 respectively).

Table 2 Shows echocardiographic results (EDD, ESD, EF) pre, after 3 and 6 months of ablation.

	Minimum	Maximum	Means ± Std. D.	*p*-value
EDD pre. Abl.	40	66	56.62 ± 5.87	
EDD after 3 months	40	62	51.08 ± 6.64	0.002
EDD after 6 months	40	56	49.23 ± 5.31	0.002
ESD pre. Abl.	24	57	41.85 ± 7.82	
ESD after 3 months	26	50	35.92 ± 7.17	0.005
ESD after 6 months	26	41	33.31 ± 4.66	0.002
EF pre. Abl.	34	52	46.69 ± 4.92	
EF after 3 months	41	71	57.15 ± 8.21	0.001
EF after 6 months	51	71	60.54 ± 5.39	0.001

EDD, end-diastolic diameter, ESD, end-systolic diameter, EF, ejection fraction, Abl., ablation.

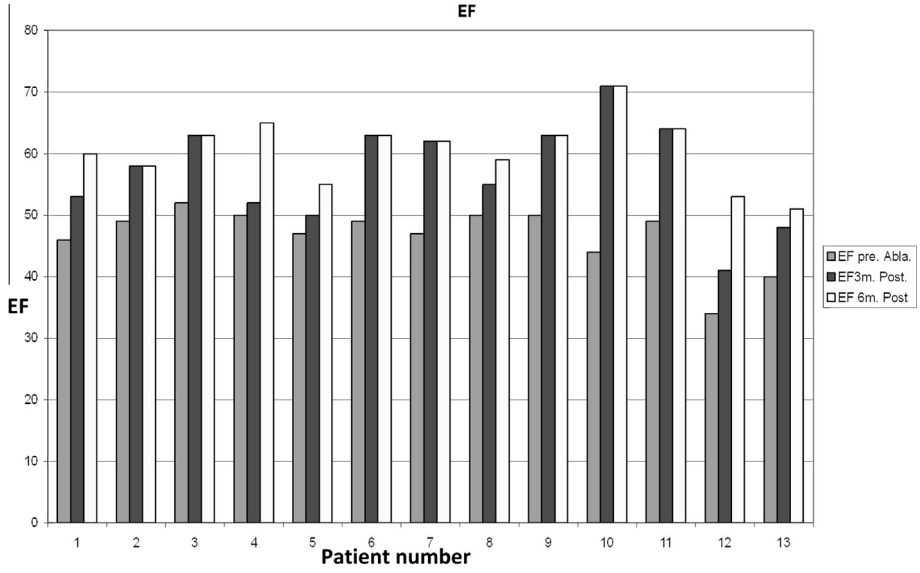

Figure 9 Shows a high significant improvement in EF 3 and 6 months following successful RFA when compared with pre-ablation EF ($p = 0.001$).

Table 3 Outcome of patients with unsuccessful RFA.

Patient no.	EDD (mm)			ESD(mm)			EF (%)			MR			NYHA class		
	0	3m	6m	0	3m	6m	0	3m	6m	0	3m	6m	0	3m	6m
1	59	62	65	46	49	51	45	42	41	III	III	III	III	III	IV
2	59	61	63	49	51	54	34	34	28	III	III	IV	III	IV	IV
3	59	57	57	46	49	49	37	31	31	III	III	III	III	III	IV

EDD, end-diastolic dimension; ESD, end-systolic dimension; EF, ejection fraction; MR, mitral regurgitation; m, month; NYHA, New York Heart Association. 0 = pre-ablation, 3m = 3 months post-ablation, 6m = 6 months post-ablation.

Study target differs from one publication to another, for example the study target for Yarlagadda et al., and betensky et al. was elimination of VPCs during the ablation and/or became noninducible with programmed electrical stimulation with or without isoprenaline infusion.[18,10] The same target was used by Takemoto et al. with the addition of nonrecurrence of PVCs for 72 h after ablation.[12] For Sekiguchi et al. the study target was reduction in PVCs to <1000 per day,[19] and for Baman et al. it was 80% reduction in PVC burden.[20]

Ge et al. studied the biggest number of cases, 553 patients with idiopathic PVCs/VT treated with RFA, 104 of them where having PVCs/VT originating from the RVOT. In his study he used PVCS <10 beats in 30 min as one of indicators of short term success of RF ablation.[11] The involvement of this indictor by Ge et al. was very helpful during determining the end point of success in our study, this is because in one of the cases of our study RF ablation diminished the frequency of PVCs dramatically but did not abolish it completely in spite of several supplementary ablation points on and around the target site, this case cannot be considered unsuccessful but with the Ge et al. indicator it was included among the successful cases. Isoprenaline administration after ablation as well as Holter recording 48 h after the procedure were used to verify whether PVCs diminution was a coincidental change in the PVCs frequency or a true actual success.

Infrequent noninducible tachycardia is a source of difficulty during RFA.[21] In our series one patient had infrequent PVCs during the procedure in spite of isoprenaline infusion, evidence of success in this case was also determined by disappearance of the PVCs after ablation with isoprenaline infusion, which was also assured by Holter recording 48 h after the procedure. If the PVCs were absent at the time of the study the patient would be canceled.

Regarding PVCs burden, we think that the use of percentage of PVCs to total beats may represent more clearly the PVCs burden than their total number; a similar number of PVCs in two patients with different number of total heart beats will have different PVCs' percentage and subsequently different PVC burdens. Some investigators used the absolute number of PVCs to define its frequency[22,23] while others used the percentage of PVCs to total heart beats[20,24] therefore we represented both results for differentiation.

At the time of initial clinical presentation our patient's initial data showed that the higher the percentage and total number of PVCs the lower the LVEF and the larger the ESD. Several studies showed that the frequency of PVCs correlates at least modestly with the extent of LV dysfunction and ventricular dilation. Patients with decreased LVEF had a higher mean PVC burden than their counterparts with normal LV function. However, there are no clear-cut points that mark the frequency at which cardiomyopathy is unavoidable. Niwano et al. used a cut point of 20 000 PVCs/day to define the high-frequency group,[22] whereas Kanei et al. used a figure of 10 000 PVCs/day.[23] Other studies defined "frequent" PVCs as $>10\%$ of total beats rather than the absolute number of PVCs[20,24] yet in some cases, a high PVC burden may not impair LV function, whereas PVC-induced cardiomyopathy can be observed in patients with lower PVC frequencies.[25] It is not known why the majority of patients with frequent PVCs have a benign course, whereas up to one third of them develop cardiomyopathy. One possible explanation is that the evaluation of PVC burden using 24-h Holter monitoring may be inadequate and may misrepresent the patient's true PVC burden.[26] Baman et al. suggested that a PVC burden of $>24\%$ had a sensitivity and specificity of 79% and 78%, respectively, in separating the patient populations with impaired versus preserved LV function.[20] Nevertheless, the majority of patients presenting with frequent PVCs had preserved LVEF as stated by some series.[6,7,22,20] Therefore, although significant, the PVC burden is not the only factor contributing to impairment of LV systolic function.[27] In our study, the LVEDD and LVESD have been reduced significantly 3 months after successful ablation ($p = 0.002$) as well as 6 months (0.005 and 0.002 respectively). The ejection fraction improved significantly after successful PVCs ablation from 46.69 ± 4.92 pre-ablation to 57.15 ± 8.21 after 3 months from successful ablation and to 60.54 ± 5.39 after 6 months from ablation ($p = 0.001$). There was also significant improvement in NYHA functional class and daily exercise capacity following ablation.

Patients in whom RFA was unsuccessful; EDD, ESD, EF, degree of mitral regurgitation by echocardiogram and NYHA functional class did not show improvement, on contraire they showed some deterioration during follow-up (Table 3). This is somewhat consistent with Bogun at al. [28] He found that abnormal LVEF in patients with frequent idiopathic PVCs normalized over a period of 6 months in 82% of patients after successful ablation (from a baseline of $34\% \pm 13\%$ to $59\% \pm 7\%$) ($p < 0.0001$) and ejection fraction declined further in patients in whom ablation was ineffective (from $34\% \pm 10\%$ to $25\% \pm 7\%$) ($p = 0.06$) during 6 months follow-up. He also observed that patients with decreased LV function had a greater PVC burden on a 24-h Holter monitor than patients with normal EF, there was also a significant inverse correlation between the PVC burden and the EF before ablation. In his control group of 11 patients with a similar PVC burden and a reduced EF who did not undergo ablation, the EF remained unchanged in 10/11 patients over 19 ± 17 months of follow-up and one patient underwent heart transplantation.[28] Bogun et al. concluded that LV dysfunction in the setting of frequent idiopathic PVCs may represent a

form of cardiomyopathy that can be reversed by catheter ablation of the PVCs.

Lelakowski et al. reported improved quality of life in 22 patients who underwent PVCs ablation. They also observed a negative correlation between PVCs load and quality of life.[29] Inspite of small number of patients in his series (4 patients with PVCs ablated from RV and 2 from LV) Taieb et al. stated that elimination of frequent isolated PVCs in patients with dilated cardiomyopathy with RFA can normalize the clinical status and LV systolic function and dimensions, regardless of the morphology or origin of the PVCs.[25]

In the present study during a mean follow up period of 13.25 ± 7.767 (6–30) months no recurrence of culprit RVOT-PVCs was observed in all patients who underwent RFA with acute successful result. This is consistent with Yamashina et al. and Van Huls van Taxis et al.,[30,17] taking in consideration the relatively short follow up period.

The average procedure time in our study was 118.12 ± 50.42 (35–200) min beginning from puncturing to pulling out the sheath. This average procedure time is comparable with Darrieux et al., who recorded an average time of 98 ± 37 min.[16]

The site of successful ablation in this study was found to be septal in ten patients (62%), free wall in three patients (19%) but not localized in the remaining three (19%) patients with failed RFA. This is consistent with many publications (septal location is more than free wall location).[5,9,31]

According to literatures approximately 10–15% of OT PVC originates from the LVOT.[32–35] In our series three cases ended unsuccessfully, this might be due to epicardial or LVOT origin of the PVCs. The ability to predict the site of origin of the PVCs from the surface electrocardiogram by Betensky's algorithm has reached specificity of 100% and sensitivity of 92%, a V2 transition ratio >0.6 predicts an LVOT origin with 91% accuracy.[10] He also stated that a PVC precordial transition occurring later than the sinus rhythm transition excludes an LVOT origin with 100% accuracy- which was the situation in the three unsuccessful patients in our study. Since the aim of our study was to define the effect of RVOT-PVC ablation on LV function and size and not testing the accuracy of the algorithms, ablating these PVCs from LVOT in those patients would be beyond the scope of this study.

7. Limitations

In our study we used bipolar pacing during pace mapping and not unipolar pacing, this would increase the surface area of the capturing electrode and lead to pacing large area of myocardium and hence to inaccuracy. However, this effect can be minimized by not using high pacing outputs and by pacing through a small interelectrode distance ($\leqslant$5 mm).[35] This was achieved by the ablation catheter that had interelectrode distance of 5 mm and with pacing without exceeding double the diastolic threshold.

8. Conclusion

The study concluded that RF catheter ablation of frequent premature ventricular complexes from right ventricular outflow tract (RVOT-PVCs) has a beneficial effect on cardiac function in patients with depressed cardiac function. It confirms a high degree of success and safety of radiofrequency catheter ablation as a curative therapy for RVOT-PVCs.

Also it confirms the positive correlation between RVOT-PVCs burden and left ventricular dimensions and function in patients without overt underlying structural heart disease.

Conflict of interest

We have no conflict of interest to declare.

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